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Influence of weight reduction on muscle performance and bone mass, structure and metabolism in obese premenopausal women

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Abstract

Muscle performance, body composition and bone mass at the lumbar spine and proximal femur with DXA, structural traits at the tibia and radius with pQCT, and biomarkers of bone metabolism were assessed at baseline and after a three-month weight reduction in obese premenopausal women. Associations between changes in weight loss and bone traits were analyzed by linear regression analysis. The mean (SD) weight loss was 4.3 (4.5) kg ranging from 14.8 kg loss to 2.1 kg gain. Muscle performance was well maintained, while no signs of bone loss or structural deterioration were observed. Changes in bone resorption were significantly associated with weight change (for CTX, $r=-0.34$; $p=0.043$, and for TRACP5b, $r=-0.35$; $p=0.032$). There were borderline ($p<0.1$) negative correlations between changes in biomarkers and bone traits. Reduced fat mass was associated with slight mean increase in cortical density of the radial shaft. Also total body BMC increased slightly. Changes in both fat and lean mass were associated with a change in BMC. Our findings suggest that mild-to-moderate weight reduction modulated bone turnover slightly, but they do not support the common notion that such a weight reduction would compromise bone rigidity, possibly partly due to well maintained muscle performance.

Keywords: Weight Reduction, Bone Fragility, Bone Loss, Bone Metabolism, Muscle Performance

Introduction

Approximately 20% of the Finnish adult population is obese (body mass index, BMI ≥ 30)[1]. Obesity is associated with obvious health risks, such as hypertension, type 2 diabetes, coronary heart disease, osteoarthritis and functional disability. In contrast, obese people are believed to have greater bone mineral density (BMD) and slower bone loss than individuals with normal body weight[2,3]. Moreover, since the common cause of hip fracture is falling, excess adipose tissue surrounding the hip may attenuate the fall-induced impact to the greater trochanter thus reducing the probability for hip fracture.

For the above reasons, one deleterious consequence of substantial weight loss could be accelerated bone loss and reduced padding of the critical anatomic sites of the body, and thus increased susceptibility to fractures in older age[4]. Should the weight reduction constitute a real problem for bone health, more effective measures during weight reduction (e.g., physical exercise, and/or dietary intervention), are needed to counter the potentially evolving bone fragility.

Several physiological explanations for reduced BMD during and after weight loss have been provided, including malnutrition, especially lack of calcium[5], and lower conversion of androgen to estrogens due to reduced mass of adipose tissue[6], or simply a decreased bone loading due to reduced body mass[7,8]. Greater body weight exerts proportionally more mechanical load on the skeleton, while different physical activities can essentially enhance the magnitude and rate of loading[9]. Some studies have shown that exercise can protect against bone loss when dieting[10-13]. Apparently, at least a

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part of this benefit is mediated through muscle work and concomitant bone loading.

An alternative explanation for an observed decrease in bone mass during weight reduction is simply methodological; i.e., the dual-energy X-ray absorptiometry (DXA) is affected by changes in soft tissues, and is thus unable to accurately measure changes in bone mass during weight loss[14, 15], let alone the inability of DXA to evaluate specifically bone structural traits[16]. Be it noted that the bone structure is the major determinant of bone fragility[17,18]. Nevertheless DXA has thus far been the major method for monitoring changes in bone mineral content (BMC) and BMD accompanying weight loss.

It has also been suggested that changes in BMD due to weight reduction are site-specific and different between predominantly trabecular bones (e.g. vertebrae) and more cortical bones[19-21]. A change in cortical-to-trabecular ratio may play an important role in determining bone strength since cortical thinning is largely responsible for reduced bone rigidity[17,18,22]. Accordingly, the DXA-based assessment of total body bone mass or density may be less sensitive to weight changes than site-specific measurements. However, the effects of weight reduction on bone mass and structure in overweight persons have scarcely been evaluated in a site-specific fashion. For this purpose, the peripheral quantitative computed tomography (pQCT) allows a feasible evaluation of bone cross-sectional geometry, cortical density, and trabecular density at the appendicular skeleton *in vivo*[23].

This study was therefore undertaken to increase our understanding on potential effects of weight reduction on bone mass, structure and metabolism. The objective was to evaluate biomechanical and metabolic influence of weight loss on bone mass and structural traits at weight-bearing and non-weight bearing bones, and to identify factors in bone metabolism that may particularly account for apparent loss of bone mass and rigidity in obese premenopausal women.

Participants and methods

Eligible participants were obese (BMI ≥30) but otherwise clinically healthy premenopausal women. Exclusion criteria were history of any severe illness or evidence for metabolic bone disease, eating disorders, menstrual irregularities, use of estrogen except for hormonal contraceptives, recent (<one year) delivery or lactation, recent (<one year) fracture/trauma and related long (>one month) immobilization, or use of other medication that could affect the skeleton or bone metabolism.

Forty obese women with mean (SD) age of 42 (7) years volunteered. At baseline their body weight was 97.1 (17.8) kg and body mass index (BMI) 35.2 (5.1). Twenty-eight women were treated for 12 weeks in weight management groups (DG) led by trained nurse practitioners in primary health care units. These groups used conservative energy restrictive diet. In addition, one weight loss group (n=12) used very-low-energy-diet (VLED) products (Cambridge Diet, Cambridgekuren, Sverige AB, Sweden) in order to attain a more substantial weight loss. The groups met weekly for 90 minutes, and the counselling topics included instructions for low-energy diets, general knowledge on diet and weight maintenance, and basics of relapse prevention techniques. All assessments were done at baseline and after the 12-week weight reduction period. Of the recruited 40 women, 37 finished the study. The study protocol was approved by the Ethics Committee of The Pirkanmaa Hospital District, and each participant gave her written informed consent prior to the intervention.

Health questionnaire

Information on self-reported diseases, injuries, medication, and diet, non-pregnant weight at age of 25, history of weight cycling, menstrual status, and lifestyle factors such as physical activity (duration, type, frequency and intensity), smoking and consumption of alcohol were obtained using a questionnaire completed with an interview.

Anthropometry and body composition

Body height was measured to the nearest 0.1 cm using a standard wall-mounted stadiometer, and body weight to the nearest 0.1 kg with a high-precision scale. Body composition (fat mass and lean soft tissue mass) was estimated with dual-energy X-ray absorptiometry (DXA, Lunar Prodigy Advance, GE Lunar, Madison, WI, USA). According to repeated measurements of 22 adults, the *in vivo* precision (coefficient of variation, CV%) was 1.3% for fat mass and 0.7% for lean mass (Sievänen, unpublished). In addition, waist circumference was measured midway between the lowest rib and superior iliac crest, and the mean of three measurements was used.

Muscle performance

The maximal isometric leg extension force was estimated by a strain gauge dynamometer at a knee angle of 110 degrees (Tamtron, Tampere, Finland), and dynamic maximal take-off force and power during a vertical countermovement jump with a force-plate (Kistler Ergojump 1.04, Kistler Instrumente AG, Winterthur, Switzerland). Functional agility was evaluated by the time taken in a figure-8 running test.

Bone measurements

Bone mineral content (BMC) of the total body, lumbar spine and left proximal femur were assessed with DXA (Lunar Prodigy Advance, GE Lunar, Madison, WI, USA). The *in vivo* day-to-day precision (coefficient of variation, CV%) is better than 1% for the lumbar spine, 1.5% for the femoral neck and 1.4% for total body BMC (Sievänen, unpublished).

	Baseline n=37	End n=37	Change	Range in change
Body composition				
Body height, cm	166.1 (6.3)			
Body weight, kg	97.5 (18.1)	93.3 (17.5)	-4.3 (4.5)	-14.8 to 2.1
BMI	35.2 (5.2)	33.7 (5.3)	-1.5 (1.7)	-5.0 to 0.8
Waist circumference, cm	107.5 (12.6)	103.7 (13.1)	-3.8 (4.3)	-15.2 to 6.5
Body fat, %	47.5 (4.5)	45.9 (5.1)	-1.6 (2.2)	-7.9 to 3.1
Body fat mass, kg	45.1 (10.9)	41.6 (10.4)	-3.5 (3.8)	-11.9 to 2.3
Body lean mass, kg	49.2 (8.6)	48.4 (8.6)	-0.8 (1.5)	-3.9 to 2.2
Muscle performance				
Isometric leg extension, N/kg	24.2 (5.2)	26.0 (5.9)	1.8 (3.0)	-5.7 to 8.5
Jumping force, N/kg	19.9 (3.2)	20.6 (3.3)	0.7 (1.8)	-2.3 to 5.8
Jumping power, W/kg	30.1 (5.2)	30.8 (5.0)	0.7 (2.3)	-6.0 to 5.4
Time of figure-8 run, s	17.0 (3.7)	16.5 (3.5)	-0.5 (0.6)	-2.3 to 0.3
Bone traits				
Total body BMC, g	2767 (333)	2812 (329)	45 (113)	-165 to 337
Lumbar spine BMC, g	69.3 (11.1)	70.1 (12.0)	0.75 (2.0)	2.8 to 5.1
Lumbar spine area, cm^2	54.9 (5.1)	55.0 (5.3)	0.14 (0.64)	-1.2 to 2.5
Femoral neck BMC, g	5.09 (0.59)	5.11 (0.59)	0.03 (0.15)	-0.37 to 0.36
Femoral neck area, cm^2	4.9 (0.3)	4.9 (0.3)	-0.03 (0.15)	-0.7 to 0.3
Trochanter BMC, g	11.9 (2.4)	11.9 (2.2)	0.04 (0.8)	-1.6 to 1.6
Trochanter area, cm^2	13.3 (1.7)	13.3 (1.5)	0.01 (0.67)	-1.3 to 1.4
Distal radius (n=36)				
TrD[1], mg/cm^3	203.8 (31.8)	203.5 (32.6)	-0.26 (4.03)	-9.1 to 7.3
BSI[2], mm^3	360.9 (68.9)	368.8 (67.0)	7.9 (18.8)	-20.9 to 53.2
CoTh[4], mm	1.82 (0.29)	1.85 (0.30)	0.03 (0.1)	-0.17 to 0.27
ToA[5], mm^2	276.3 (42.3)	274.4 (40.9)	-1.9 (9.7)	-16.5 to 19.7
Radial shaft (n=36)				
CoD[3], mg/cm^3	1203.0 (19.1)	1203.3 (20.0)	0.34 (9.78)	-27.1 to 19.3
BSI[2], mm^3	211.0 (38.4)	213.5 (38.9)	2.5 (7.2)	-8.4 to 20.7
CoTh[4], mm	2.75 (0.42)	2.75 (0.40)	-0.01 (0.14)	-0.36 to 0.36
ToA[5], mm^2	103.8 (13.0)	104.1 (12.8)	0.3 (2.6)	-6.2 to 6.3
Distal tibia				
TrD[1], mg/cm^3	240.6 (27.7)	241.3 (27.1)	0.65 (2.24)	-3.3 to 8.3
BSI[2], mm^3	1361.4 (279.3)	1348.6 (298.1)	-12.8 (55.5)	-177.8 to 61.9
CoTh[4], mm	2.00 (0.50)	2.01 (0.52)	0.01 (0.09)	-0.27 to 0.20
ToA[5], mm^2	870.2 (138.9)	872.6 (134.1)	2.4 (14.1)	-27.5 to 29.2
Tibial shaft				
CoD[3], mg/cm^3	1145.6 (20.9)	1145.1 (20.6)	-0.45 (5.63)	-12.7 to 11.1
BSI[2], mm^3	1928.1 (294.4)	1938.5 (301.4)	10.4 (50.9)	-77.3 to 169.6
CoTh[4], mm	7.04 (0.62)	7.03 (0.63)	0.01 (0.10)	-0.33 to 0.25
ToA[5], mm^2	468.6 (49.8)	468.9 (50.1)	0.4 (4.1)	-8 to 9.5
Bone metabolism				
CTX, nM	10.8 (10.7)	11.4 (7.1)	0.60 (7.46)	-31.5 to 17.3
TRACP 5b, U/L	1.5 (0.4)	1.4 (0.5)	-0.12 (0.38)	-0.86 to 1.16
PINP, µg/L	35.6 (15.9)	33.9 (13.0)	-1.73 (11.77)	-37.1 to 24.8
Calcium intake, mg/d	888 (271)	940 (341)	52 (271)	-362 to 753

[1]TrD=Trabecular density; [2]BSI=Bone strength index; [3]CoD=Cortical density; [4]CoTh=Cortical thickness; [5]ToA=Cross-sectional area

Table 1. The mean (SD) body composition, muscle performance, bone traits and bone metabolism at baseline and the end of the 3-month weight reduction period, and the mean of absolute change (SD) and range in changes in premenopausal women.

In addition to DXA measurements, left radius and tibia were scanned with peripheral quantitative computed tomography (pQCT; XCT 3000 Stratec Medizintechnic GmbH, Pforzheim, Germany). The tomographic slices were taken from the shaft and distal part of the tibia (50% and 5% from the distal endplate of the tibia, respectively) and radius (30% and 4% from the distal endplate the radius, respectively) according to our standard procedures[23]. For the shaft sites, cortical density (CoD, g/cm^3) and density-weighted polar section modulus (BSI, an index of bending strength) were assessed. For the distal sites, trabecular density (TrD, g/cm^3) and BSI were assessed. In our laboratory, the *in vivo* precision of the pQCT measurements ranges from 0.7% (tibial shaft CoD) to 7.7% (distal radius BSI)[23]. One radius pQCT scan was excluded from the analyses because of clear movement artefact during the baseline measurement.

As serum markers of bone turnover, tartrate-resistant acid phosphatase isoforms 5b (TRACP5b)[24] and C-terminal cross-linked telopeptides of type I collagen (CTX) were determined as a marker of bone resorption, and amino-terminal propeptide of type I procollagen (PINP) as a marker of bone formation[25].

Venous blood samples were obtained after 12-hour fasting at baseline, and at 12 weeks. Serum was separated by centrifugation, aliquoted and stored at -70˚C until analyses. The bone markers were determined using commercial immunoassays. PINP was determined using UniQ™ PINP assay (Orion Diagnostica, Espoo, Finland) and CTX with CrossLaps® assay (IDS Ltd, Boldon, UK) and TRACP 5b with BoneTRAP® assay (IDS Ltd). Analytical and biological coefficient of variations (CV%) for PINP were 3.6% and 17.9%, for TRACP5b 2.1% and 12.5% and for CTX 2.6% and 22.4%, respectively[26].

Dietary intake

Calcium intake was estimated at baseline and at the end of the intervention using a 7-day calcium intake dietary and calculated by Micro-Nutrica software (Social Insurance Institution, Helsinki, Finland)[27].

Statistical analysis

Means and standard deviations (SD) were used as descriptive statistics. Paired sample t-test was used to test changes between baseline and 12-week measurements. Pearson's product-moment correlation coefficients were used to describe the associations between weight changes and changes in muscle performance, bone traits and biomarkers. Linear regression analysis was used to analyze associations of weight change with changes in bone traits. The associations were adjusted for age. Changes in fat mass and lean mass were used as potential predictors for changes in bone traits.

Results

Table 1 shows the baseline data in body composition, bone traits and biomarkers of bone metabolism, and their changes after the 12-week weight reduction period. The mean (SD) decrease in body weight was 4.3 (4.5) kg with individual range from 14.8 kg loss to 2.0 kg gain, while no signs of bone loss or structural deterioration were observed. The mean decrease in waist circumference was 3.8 (4.3) cm with a range from 15.2 cm decrease to 6.5 cm increase. The mean declines in fat mass and lean mass were 3.5 (3.8) kg with a range from 11.9 kg decrease to 2.3 kg increase, and 0.8 (1.5) kg ranging from 3.9 kg decrease to 2.2 kg increase, respectively. Mean time in physical activity was 2.4 (1.9) hours per week walking being the most common recreational activity. The participants did not report any changes in physical activity.

Table 1 shows also baseline data and changes in muscle performance. Mean (95% confidence interval) increase in relative isometric leg extension force was 1.83 (0.80 to 2.85) N/kg. Also the relative jumping force and power improved, although not statistically significantly ($p=0.083$ and $p=0.060$ for muscle force and power, respectively). The time taken in figure-8 run was reduced significantly by 0.48 (0.28 to 0.69) s.

Correlations between changes in body composition, muscle performance, biomarkers of bone metabolism and bone traits in premenopausal women are shown in Table 2. Changes in resorption markers CTX and TRACP5b correlated with weight change ($r=-0.34$, $p=0.043$ and $r=-0.35$, $p=0.032$, respectively), while the change in the formation marker PINP did not reach statistical significance ($r=-0.27$, $p=0.11$). There were borderline negative correlations between changes in PINP and CoD of the tibial shaft ($r=-0.29$, $p=0.083$), as well as between TRACP5b and BSI of the tibial shaft ($r=-0.29$, $p=0.082$). There was no correlation between muscle performance and PINP, while the correlations were positive between changes in CTX and isometric leg extension force ($r=0.379$, $p=0.023$), and TRACP5b and jumping force ($r=0.433$, $p=0.009$) and jumping power ($r=0.336$, $p=0.049$).

Correlations between changes in weight and bone traits were inverse and of borderline significance: for distal radius TrD, $r=-0.34$, $p=0.046$, and for radial shaft CoD, $r=-0.32$, $p=0.059$ (Table 2). On individual basis, those women with the greatest weight loss seemed to show slight mean increase in radial or tibial CoD, while women with a small reduction in weight seemed to have no bone change on average (Figure 1). The weight loss was not associated with a change in bone strength index (BSI). Calcium intake at baseline or change in calcium intake during weight reduction was not associated with changes in bone traits. Similarly, physical activity was not associated with changes in bone variables.

Both fat mass and lean mass were independent predictors for the change in total body BMC. The β-coefficient (95% CI) for changes in fat and lean mass were 12.0 (3.0 to 21.0) and -37.2 (-59.9 to -14.5), respectively. This means that while total body BMC increased slightly during the intervention, every one kg loss in fat mass was associated with a 12 g smaller mean increase in BMC, and one kg loss in lean mass was associated with a 37.2 g greater mean increase in total body BMC. Compared to the change in lean mass, the change in fat mass was also a better predictor for changes in radial

Changes	Weight n=37	CTX n=37	TRACP-5b n=37	PINP n=37
Body weight	1	-0.34*	-0.35*	-0.27
Waist circumference	0.87 ***	-0.26	-0.32	-0.21
Body fat	0.94 ***	-0.25	-0.28	-0.21
Lean body mass	0.49 **	-0.33*	-0.35*	-0.17
Muscle performance				
Isometric leg extension	-0.32	0.38**	0.15	0.010
Jumping force	-0.29	0.15	0.43**	0.09
Jumping power	-0.34*	0.003	0.34*	0.22
Time of Figure-8 run	0.16	-0.28	-0.24	0.28
BMC				
Total body BMC	0.16	0.06	-0.03	-0.06
Femoral neck BMC	-0.19	0.02	-0.16	-0.23
Trochanter BMC	0.20	-0.22	-0.19	-0.21
Lumbar spine BMC	-0.23	0.09	-0.14	-0.23
Distal tibia				
TrD[1]	-0.04	0.24	-0.07	0.004
BSI[2]	0.18	0.03	-0.29	0.09
Tibial shaft				
CoD[3]	-0.27	0.05	0.16	-0.29
BSI	0.01	-0.02	-0.07	-0.02
Distal radius, n=36				
TrD[1]	-0.34*	-0.03	-0.08	-0.06
BSI[2]	0.20	-0.26	-0.25	-0.34*
Radial shaft, n=36				
CoD[3]	-0.32	-0.12	0.02	-0.22
BSI[2]	-0.18	0.25	-0.06	-0.12

**p<0.05; **p<0.01; ***p<0.001; [1]TrD=Trabecular density; [2]BSI=Bone strength index; [3]CoD=Cortical density*

Table 2. Correlations between changes in body composition, muscle performance, biomarkers of bone metabolism and bone traits in premenopausal women.

TrD, one kg decrease in fat mass was associated with a marginal 0.4 mg/cm^3 mean increase in trabecular density. Similarly, there was an association between changes in fat mass and CoD of the radial and tibial shafts (Table 3).

Discussion

We did not find any detrimental influence of weight reduction, at least in short-term, on bone mass or structure during the period of a 3-month study among obese, but otherwise healthy premenopausal women. Muscle performance was well maintained, even somewhat improved during weight reduction. Also, the correlation between weight change and changes in trabecular density at the distal radius, and cortical density at the radial and tibial shafts were negative indicating that those women with the greatest weight loss appeared to have some increases in these bone traits. On the other hand, it is stressed that all observed bone changes were generally marginal (e.g. for total body BMC 1.7% and for radial shaft CoD 0.03%), and apparently had no effect on bone rigidity to either direction.

Increased bone turnover is commonly linked to weight reduction[28]. In the present study the observed mean changes in bone turnover remained so small that our results do support this notion, but argues our above interpretation of no detrimental effect on bone. Regarding potential factors that may affect bone turnover during weight loss, high calcium intake has been suggested to suppress the elevated rate of bone resorption[29-31]. Similarly, physical training has been found to protect against increased bone turnover[10,32]. However, results on the effects on bone mass are conflicting. Weight loss, even when combined with exercise, results in a decrease in hip BMD among obese older adults[33]. Resistance training during weight loss also did not have any effect on BMD changes among premenopausal women[34]. Although our participants did not report any changes in level of physical activity during the dieting, weight reduction was associated with improvements in relative muscle performance, as judged from some improvements in functional agility, muscle force and power. Lean mass is more strongly associated with bone mass and strength than fat mass[35-37], and due to reduced body weight, it is apparent that muscle force and power can be more efficiently employed during physical activity, and the prevalent bone loading maintained. Mean

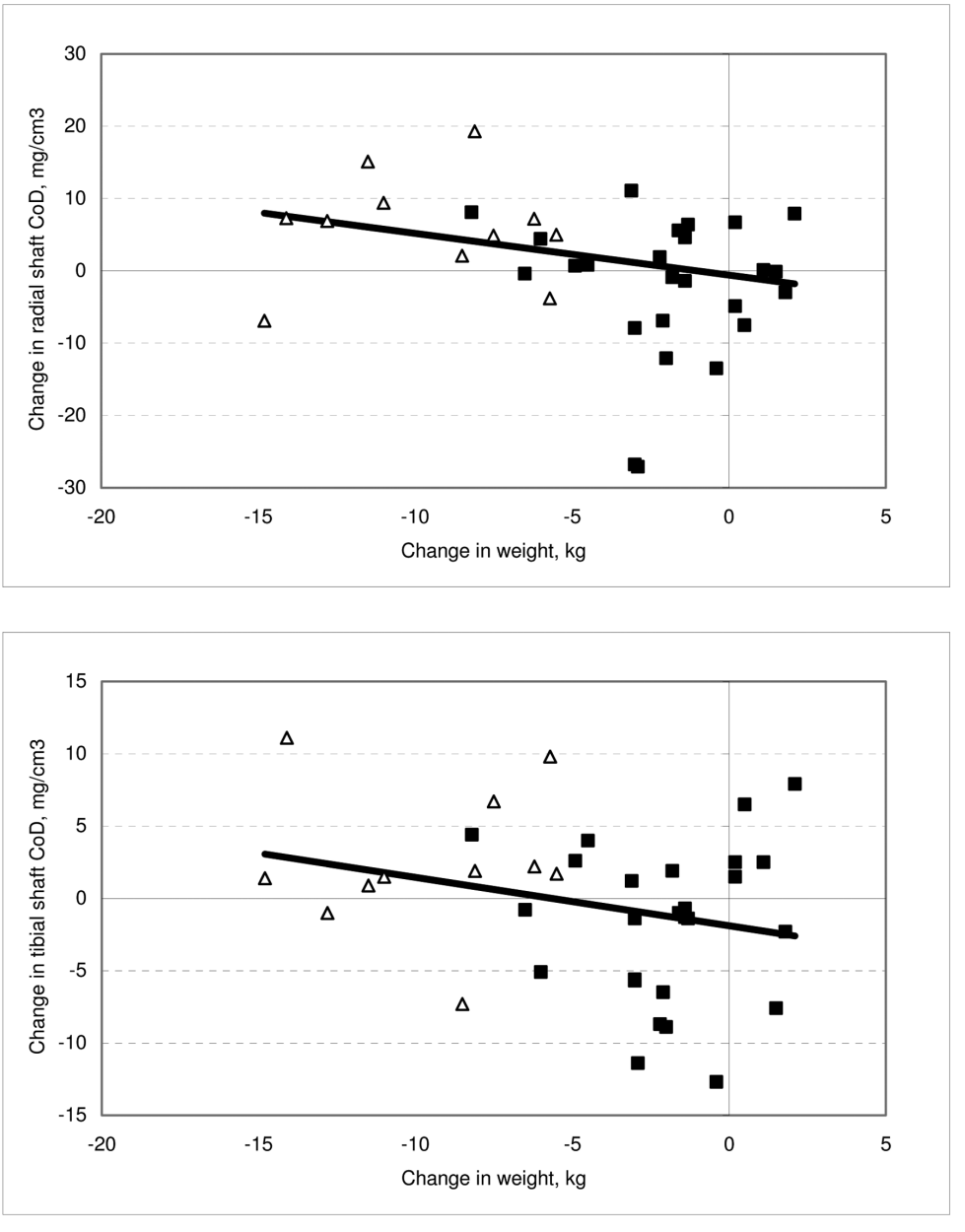

Figure 1. Association and regression lines between changes in body weight and cortical density at the radial shaft (R^2=0.11; SEE=9.50 mg/cm^3, y=-2.58 -0.34 * x) and at the tibial shaft (R^2=0.07; SEE=5.51 mg/cm^3, y=-1.89-0.34* x). Open triangles denote the individual changes in the VLED group and solid squares denote the changes in the DG group (i.e. those on the conservative treatment).

calcium intake was adequate among our subjects at baseline and was also well maintained during the weight loss period.

Possible explanations for observed positive correlations between weight reduction and bone loss in earlier studies may pertain to magnitude of weight loss *per se*, or inconsistencies in DXA measurements. In our earlier 3-month weight reduction intervention with subsequent 9-month walking training and 24-month follow-up in obese premenopausal women, a small but statistically significant decline in radius BMD during weight reduction was observed, but during the 2-year follow-up an almost total regain in body weight was accompanied by a similar regain in BMD. However, spinal BMD did not seem to recover; not even in those participants who gained more weight than they lost[21]. In the current study we did not find such deterioration in bone traits at all. This may be due to more modest weight loss in this study, which, however, is comparable with several earlier reports[12,38-40]. Regarding the DXA system, the device used in this study represented a different brand than in our previous study, and it may be so that brand-specific

Bone site	β-coefficient	SE	P-value
Change in total body BMC			
Constant	43.27		
age at baseline, y	0.342	2.52	0.89
change in fat mass, kg	11.99	4.57	0.013
change in lean mass, kg	-37.22	11.60	0.003
R^2=0.29, SEE=99.3 g, n=37			
Change in distal radius trabecular density			
Constant	-3.797		
age at baseline, y	0.049	0.099	0.62
change in fat mass, kg	-0.400	0.179	0.032
change in lean mass, kg	-0.021	0.454	0.96
R^2=0.15, SEE=3.89 g/cm^3, n=36			
Change in radial shaft cortical density			
Constant	6.644		
age at baseline, y	-0.221	0.217	0.32
change in fat mass, kg	-1.351	0.393	0.002
change in lean mass, kg	2.175	0.997	0.037
R^2=0.30, SEE=8.53 g/cm^3, n=36			
Change on tibial shaft cortical density			
Constant	9.373		
age at baseline, y	-0.266	0.123	0.038
change in fat mass, kg	-0.694	0.223	0.004
change in lean mass, kg	1.277	0.567	0.031
R^2=0.32, SEE=4.85 g/cm^3, n=37			

Table 3. Regression models for changes in bone mass and density (only models with one or more significant predictors are included).

calibration and assumptions employed in the data processing and analysis may partly account for inconsistent findings. On the other hand, a recent report of Redman et al. supports our current results[41]. In that study, compared with the weight-maintained control group, the weight loss groups, with more than 10% weight loss after a six-month period, showed no significant changes in bone mass, while some changes in serum bone markers were observed[41].

DXA-based body composition analysis enables the division of the soft tissue body mass into fat and lean components. When comparing the effects of fat and lean tissue separately on bone traits, fat mass appeared to be somewhat stronger predictor for changes at the radius than lean mass. However, the association was not consistent for all measured bone traits or sites, since lean mass was associated with increased total body BMC, and fat mass was associated with a smaller mean increase. Besides being marginal in general, these associations must be interpreted with caution because the bone results may be strongly confounded by inherent inaccuracies in the DXA method, those arising from changes and disparities in soft tissue[42,43]. Furthermore, individual changes in soft tissue anthropometry were large, and distribution and proportion of fat vs. lean tissue in the local bone sites could not be analyzed. It should be pointed out that the present pQCT measurements, not being compromised by similar soft tissue related inaccuracies as DXA is, indicated no signs of significant loss or deterioration in any trabecular or cortical trait at the non-weight bearing radius or weight bearing tibia.

Our study had some limitations. First, the modest mean weight loss may be considered inadequate in terms of potential effect on bone. On the other hand, the individual variation was large, which enhanced the correlation analysis. In fact, this large variation was a predetermined aim in the study protocol. For this reason, the two different weight reduction programs (DG and VLED) were included in the study without intention to compare between-group results. Second, the duration of the weight reduction intervention was 12 weeks, which may be a short period to detect changes in DXA or pQCT-measurements. However, a three-month period is enough to detect changes in bone metabolism, and losses in bone mass can be seen in three months should such changes occur[44,45]. In other weight reduction studies, significant declines in the hip and lumbar spine BMD have been observed in three months both in postmenopausal women[10,46] and premenopausal women[21]. Third, lifestyle factors were not measured but obtained by interview. Whereas the participants did not report any changes in their habitual physical activity, it is possible that at least those who were successful in dieting had increased their habitual activity. If so, more specific measurements could have captured such changes in physical activ-

ity (e.g. in terms of intensity)[47] and helped us to explain our findings further. Despite participating in the weight reduction program, some participants did not lose weight, or some of them even gained weight. Obviously, they did not follow the dieting instructions. Since the participants did not keep food records during the dieting, it was not possible to estimate their energy intake or intakes of specific nutrients except calcium. Calcium intake was well maintained during the study period at least at the group level. With regard to the estimation of energy intake, obese people are known to underestimate their food intake even in normal circumstances[48]. Given this inherent methodological uncertainty, the food records were found unfeasible for this study and thus not used.

In conclusion, our findings suggested that modest reductions in body weight and fat tissue can modulate bone turnover to some extent. However, our results do not support the common notion that modest weight loss compromises bone rigidity, at least not among premenopausal women. It is possible that well-maintained muscle performance accounted for this.

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